REMARKS

In the Official Action dated September 21, 2007, Claims 11, 13 and 15 are pending and under consideration on the merits. Claims 11 and 15 are rejected under 35 U.S.C. §102(e) as allegedly anticipated by Ghosh et al. (U.S. Patent No. 6,268,398) with evidence by Lang et al. (U.S. Patent Publication 2005/0064501). Claims 11, 13 and 15 are rejected under 35 U.S.C. §103(a) as allegedly unpatentable over Ghosh and in view of Thiam et al. (*FEBS Letter*, 459:285-90, 1999) with evidence by Lang et al.

This Response addresses each of the Examiner's rejections. Applicant therefore respectfully submits that the present application is in condition for allowance. Favorable consideration of all pending claims is therefore respectfully requested.

Claims 11 and 15 are rejected under 35 U.S.C. §102(e) as allegedly anticipated by Ghosh et al. (U.S. Patent No. 6,268,398) with evidence by Lang et al. (U.S. Patent Publication 2005/0064501).

The Examiner acknowledges that Ghosh et al. do not teach a method of causing amnesia in an animal as claimed by the present application. However, the Examiner alleges that Ghosh et al. teach a method of administering chelerythrine as a kinase inhibitor for therapy of Alzheimer's disease, diabetes mellitus, neuropathy, epilepsy, stroke and traumatic injury to brain. The Examiner contends that these diseases would have pain syndrome associated with the diseases. The Examiner contends the administration of chelerythrine inherently has the amnesiac effect. In addition, the Examiner alleges that Lang et al. teach that the chelerythrine suppresses the activation of the Na+ channel. As such, the Examiner asserts that Lang et al. evidence that chelerythrine can decrease synaptic transmission. The Examiner also alleges that Lang et al. teach treatment of epileptic seizures with kinase inhibitors.

In the first instance, Applicant respectfully submits that as indicated in Applicant's previous response dated March 16, 2006, the reference to Lang et al. does not qualify as a reference under 35 U.S.C. §102(e). Applicant also observes that the Examiner in the present Official Action uses the Lang et al. reference as evidence in support for allegations based on Ghosh et al., which is the §102(e) reference relied upon by the Examiner.

However, as discussed in the previous response, Applicant respectfully submits that the reference to Ghosh et al. teaches away from the reference. As such, regardless of any alleged supporting evidence of Lang et al., Ghosh et al. do not teach or suggest a method of causing amnesia in an animal in any event.

As argued previously, Ghosh et al. is directed to compositions and methods for treatment of certain mitochondria-associated diseases such as cancer, psoriasis, stroke, Alzheimer's disease and diabetes. Ghosh et al., disclose that "[d]efective mitochondrial activity . . . may result in . . . (iv) the release of factors . . . that initiate or stimulate the apoptosis cascade." See col. 1, lines 44-55. Ghosh et al. disclose that "[a] number of diseases and disorders are thought to be caused by or be associated with alterations in mitochondrial metabolism and/or inappropriate induction or suppression of mitochondria-related functions leading to apoptosis." See col. 1, lines 56-59 (emphasis added). Ghosh et al. further suggest that Alzheimer's disease may be caused by apoptotic cell death. As such, with respect to neurological diseases or disorders, such as Alzheimer's disease, Ghosh et al. imply suppressing apoptosis.

In this regard, Applicant observes that Ghosh et al. teach that disorders, such as cancer, involve the unregulated and undesirable growth (hyperproliferation) of cells that escape a mechanism that normally triggers apoptosis in such undesirable cells. See col. 7, lines 54-57. As such, with respect to diseases like cancer, Ghosh et al. suggest inducing or triggering apoptosis

by certain compounds. To this end, Applicant observes that Ghosh et al. mentions that chelerythrine can be one of such compounds (or "apoptgens") for use to induce apoptosis. See col. 22, lines 18-35. Applicant also observes that the above-mentioned reference to chelerythrine appears to be the only time Ghosh et al. refer to chelerythrine.

As such, Applicant respectfully submits that Ghosh et al. do not teach chelerythrine as a kinase inhibitor for treating Alzheimer's and other neurological diseases. In fact, according to Ghosh et al., chelerythrine may cause Alzheimer's disease by inducing apoptosis. Accordingly, Applicant submits that Ghosh et al. teaches away from the claimed invention.

Therefore, in view of the foregoing and contrary to the Examiner's allegation, Applicant submits that Ghosh et al. do not teach a method of administering chelerythrine as a kinase inhibitor for causing amnesia in an animal or decreasing synaptic transmission in animals suffering Alzheimer's disease, diabetes mellitus, neuropathy, epilepsy, stroke and traumatic injury to brain. In fact, as discussed above, the teaching of Ghosh et al. suggests that chelerythrine may cause or worsening the above-listed diseases. Given that Ghosh et al., which is the reference relied upon by the Examiner under §102(e), teach away from the present invention, the evidence, if any, provided by Lang et al. in support of Ghosh et al. is meaningless.

Therefore, the rejection of Claims 11 and 15 under 35 U.S.C. §102(e) as allegedly anticipated by Ghosh et al. as evidenced by Lang et al. is overcome and withdrawal thereof is respectfully requested.

Claims 11, 13 and 15 are rejected under 35 U.S.C. §103(a) as allegedly unpatentable over Ghosh and in view of Thiam et al. (*FEBS Letter*, 459:285-90, 1999) with evidence by Lang et al.

Applicant respectfully submits that Thiam et al. merely teach that the distribution of palmitoylated modified PKC-ζ pseudosubstrate lipopeptides is possibly correlated with a selective induction of apoptosis. Nowhere do Thiam et al. teach a method of causing amnesia or decreasing synaptic transmission by administering a therapeutically effective amount of a PKM-ζ inhibitor, as claimed.

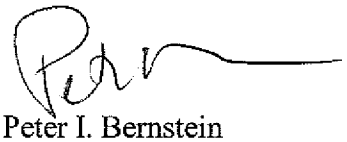
Applicant submits Ghosh et al. do not teach or suggest the method of the present invention as discussed above. As discussed above, the primary reference to Ghosh et al. in fact leads the skilled artisan away from the present invention. The secondary reference to Thiam et al. and does nothing to ameliorate the deficiencies of Ghosh et al.

As such, Applicant respectfully submits that the combination of the cited art cannot achieve the present invention. Moreover, Claim 13, as previously presented, no longer depends on Claim 11. Nowhere does the cited art teach or suggest the subject matter of Claim 13, which is directed to a method of causing amnesia or decreasing synaptic transmission in an animal suffering from a traumatic stress disorder, a phobia, a pain syndrome or epilepsy comprising the administration of a therapeutically effective amount of a PKMζ inhibitor to said animal, wherein said PKMζ inhibitor is myristolated zeta inhibitory pseudosubstrate peptide.

Therefore, the rejection of Claims 11, 13 and 15 under 35 U.S.C. §103(a) as allegedly unpatentable over Ghosh et al. in view of Thiam et al. as evidenced by Lang et al. is overcome and withdrawal thereof is respectfully requested.

In view of the foregoing amendments and remarks, it is firmly believed that the subject application is in condition for allowance, which action is earnestly solicited.

Respectfully submitted,



Peter I. Bernstein
Registration No. 43,497

Scully, Scott, Murphy & Presser, P.C.
400 Garden City Plaza, Suite 300
Garden City, New York 11530
(516) 742-4343
PIB/ZY:dg